seedlings. A nodule was removed for histopathological examination from the region of the free margin of the lesser omentum.

In the post-operative period the patient developed a thrombosis of the right calf. This responded to anticoagulant therapy, and he left hospital for convalescence four weeks after admission.

Pathological Report (Dr. A. G. Stansfield).—"Nodule from free edge of lesser omentum. Small lobulated mass of adipose tissue containing two soft red vascular nodules, the larger 6 mm. in diameter. Microscopically both nodules show the characteristic of splenic tissue, displaying typical Malpighian bodies and venous sinuses separated by Billroth cords. Confirmation of the nature of the tissue is provided by the reticulin pattern in a silver impregnation preparation. The latter shows quite clearly the ladder-like structure of the reticulin fibres in the walls of the venous sinuses. Polymorphonuclear neutrophil leucocytes are present in considerable numbers both in the sinuses and in the pulp cords.

FIG. 1.—Note fibrous capsule, Malpighian bodies, separated by Billroth cords, and venous sinuses—characteristic structure of splenic tissue. (Haematoxylin and eosin. $\times 100$.)

FIG. 2.—Note the ladder-like structure of the reticulin fibres on the walls of the venous sinuses. (Silver impregnation preparation. $\times 450$.)

The nodules are not enclosed by a normal splenic capsule, but there is a more or less distinct fibrous capsule at the periphery which incorporates focal aggregations of lymphoid tissue and in places joins up with surviving remnants of splenic trabeculae. The free surface of the nodule is covered by a continuous layer of mesothelium. The appearance of these nodules suggests that they represent fragments of spleen implanted in the peritoneum."

Summary and Conclusions

Splenosis, or autotransplantation of the splenic tissue, may follow rupture of the spleen. This is a rare condition and fewer than 20 cases have been described as a laparotomy finding.

If one is not familiar with the condition it may be mistaken for endometriosis, multiple angiomas, or carcinomatosis. Although usually symptomless in itself, it has produced small-bowel obstruction through adherence of loops of jejunum.

Elective splenectomy should be performed as carefully as possible to prevent accidental rupture, with later recurrence of the disease—for example, congenital haemolytic jaundice—due to autotransplantation of splenic tissue.

A case of splenosis is reported in which the condition was found at laparotomy for haematemesis. In such a case the possibility of confusion with angiomatosis of intestine is mentioned.

My thanks are due to Mr. Rodney Maingot, under whose care the case was admitted, for his help and advice in the surgical management and in the preparation of this paper, and also to Dr. Alfred Stansfield for his painstaking and thorough examination of the specimen removed.

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SYSTEMIC LUPUS ERYTHEMATOSUS PRESENTING WITH SULPHONAMIDE HYPERSENSITIVITY REACTION

BY

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The clinical picture of systemic lupus erythematosus (S.L.E.) is now well known and there have been numerous descriptions in the literature (Baehr *et al.*, 1935; Klemperer *et al.*, 1941; Montgomery and McCreight, 1949; Gold, 1951a; Cohen and Cadman, 1953; Dubois, 1953; Gold and Gowing, 1953; Jessar *et al.*, 1953; Harvey *et al.*, 1954; Baehr, 1955); the pathological changes too have been often described (Klemperer *et al.*, 1941; Harvey *et al.*, 1954). Treatment with cortisone or corticotrophin (A.C.T.H.) is the first measure which has been shown to exert a significant effect on the course of the disease, though it is less certain how it affects the ultimate prognosis (Carey *et al.*, 1950; Soffer *et al.*, 1950, 1952, 1954; Haserick *et al.*, 1951; Dubois *et al.*, 1952; Johnson and Meyer, 1952; Shearn and Pirofsky, 1952; Haserick, 1953; Harvey *et al.*, 1954).

On the other hand, the aetiology of S.L.E. remains obscure. Three factors seem to be concerned: constitutional factors, including sex and age, are

thought to determine the peculiar type of reaction to the noxious agent; the disease process is set in motion by a hypersensitivity reaction to drugs, sera, or bacteria; and, finally, sunlight and possibly other agents may provoke an exacerbation or induce "systematization" of a disease previously confined to the skin.

The evidence that S.L.E. is a manifestation of hypersensitivity is fairly good, but by no means conclusive; it can be summarized thus:

1. The main histological feature of S.L.E., fibrinoid necrosis of collagen, occurs also in other diseases, such as acute rheumatism and polyarteritis nodosa, which are believed to be hypersensitivity diseases (Rich, 1946). However, fibrinoid necrosis is also found in other unrelated conditions, such as acute bacterial infections, chemical or mechanical injury (including malignant hypertension), in the base of peptic ulcers, and in acute pancreatitis (Klemperer, 1948). S.L.E. has other pathological and clinical features in common with polyarteritis nodosa, acute rheumatism, and serum sickness (Rich, 1946).

2. Lesions resembling those of S.L.E. can be demonstrated in animals with experimentally induced hypersensitivity to serum (Rich, 1946; Rich and Gregory, 1947). Fox (1943) describes the case of a patient in whom S.L.E. started with a hypersensitivity reaction to horse serum.

3. The improvement brought about by cortisone, an agent known to modify allergic responses, dramatically often in acute cases, suggests an allergic basis for the disease. However, it must be admitted that cortisone is sometimes effective in diseases not usually regarded as allergic and is capable of modifying the tissue reaction to a variety of injurious agents.

4. Acquired auto-immune haemolytic anaemia (Michael *et al.*, 1951; Dubois, 1952), associated with circulating antibody and a positive Coombs test, and thrombocytopenic purpura (Michael *et al.*, 1951; Conley, 1952; Harvey *et al.*, 1954) occur in a proportion of cases of S.L.E. The occurrence of increased plasma gamma-globulin (Coburn and Moore, 1943; Flynn, 1954) and biological false-positive reactions—for example, in serum tests for syphilis (Haserick and Long, 1952)—also suggests a disturbance of antibody production. Some patients with S.L.E. show a remarkable ability to form multiple antibodies to transfused blood (Callender *et al.*, 1945).

5. Systematization of chronic discoid L.E. and exacerbations of S.L.E. have often been observed to follow the administration of various drugs to which the patients appear to be hypersensitive.

6. In most cases micro-organisms have not been demonstrated in the lesions or blood stream, though occasionally terminal bacterial septicaemias have been observed.

Role of Sulphonamides

Sulphonamide hypersensitivity (drug fever and skin rashes) is well recognized and has frequently been described; fatal cases have occurred and lesions resembling those seen in serum sickness are reported (Hawking and Lawrence, 1950). Rich (1942a, 1942b) has observed lesions resembling those of polyarteritis nodosa at necropsy in patients treated with serum or sulphonamides, or both, who died shortly after the onset of a hypersensitive reaction.

It has been repeatedly observed that various drugs and antibiotics, notably sulphonamides, may provoke dissemination or systematization of chronic discoid L.E., or exacerbation of the systemic form of the disease (Gold, 1951b; Harvey *et al.*, 1954). It is less certain that sulphonamides can ever induce L.E. in a previously healthy patient showing no evidence of cutaneous or systemic L.E. Harvey *et al.* (1954), in their Table II, refer to the development of allergic manifestations to sulphonamide before the diagnosis of L.E., but do not state whether there were already other symptoms or signs attributable to L.E.

Gold (1951b) describes five cases (Nos. 1, 4, 6, 7, and 8) in which sulphonamides had been given before the diagnosis of S.L.E. was made. In Case 1 facial discoid L.E. appeared in 1936, and not until 1947 were sulphonamides first administered; in Case 6 thrombocytopenic purpura preceded the administration of sulphonamides; and in Case 7 the first course of sulphonamide was prescribed for facial L.E. and only later did the patient manifest hypersensitivity to sulphonamide and develop frank S.L.E. Only Cases 4 and 8 had had sulphonamides before the development of any sign or symptom attributable to L.E.; but in neither of these cases was sulphonamide administration related closely in time to the onset of L.E., nor was there evidence of abnormal sensitivity to sulphonamides before the development of other signs of L.E.

Gold (1951b) reviews reports of cases of cutaneous L.E. apparently benefited by sulphonamide treatment, which may act by eradicating a septic focus with its bacterial antigens. He emphasizes that it is when prolonged or repeated courses are given that the sulphonamide may itself act as a provoking antigen.

In the following case fatal S.L.E. followed shortly after a severe episode of sulphonamide hypersensitivity in a patient suffering from bronchiectasis to whom repeated courses of sulphonamide had been given.

Case Report

A hospital clerk was first seen in 1948 (when aged 16). She gave a history of productive cough since pneumonia at 1 year. Bronchography showed bilateral bronchiectasis. In 1949 an episode of right-sided pleurisy was treated with a short course of a sulphonamide and she was admitted to hospital with oliguria and haematuria, from which she recovered without active treatment. Following this a persistent urinary infection was treated with mandelic acid and "sulphatriad." She remained well, but with persistent productive cough (treated by postural drainage) until January, 1952, when a cold was followed by right-sided pleurisy and an increase in her cough and sputum. Vigorous physiotherapy produced no improvement, and on February 18 she was admitted to hospital.

On examination her temperature was 99.6° F. (37.6° C.); there was no clubbing; widespread rhonchi and bilateral basal rales were heard. She was producing 1 oz. (28 ml.) of mucopurulent sputum daily. Radiography showed peribronchial changes in both lower zones. The white-cell count was 3,300 per c.mm. (polymorphs 50%, lymphocytes 37%, monocytes 13%). Intramuscular penicillin and oral sulphadimidine (1 g. t.d.s.) caused rapid improvement; her temperature fell to normal and the rales disappeared. On February 28 a morbilliform rash developed and her temperature was 100.8° F. (38.2° C.); mepyramine maleate (100 mg. t.d.s.) was started. Next day the rash had disappeared, but the fever persisted; penicillin and sulphadimidine were stopped. On March 4 the spleen tip was palpable and there were enlarged lymph nodes on the left side of the neck and in the right axilla. The white-cell count was 3,200 (polymorphs 72%, lymphocytes 22%, monocytes 5%, eosinophils 1%); there were no atypical cells. The Paul-Bunnell test was negative on two occasions. The temperature finally returned to normal on March 11.

On March 17 penicillin and sulphadimidine were restarted, with mepyramine maleate, in preparation for a bronchogram. The rash, lymphadenopathy, and fever returned immediately (her temperature was 103.8° F. (39.9° C.) at 10 p.m. on March 17). The antibacterial drugs were discontinued on March 19, and the next day her temperature was normal. Bronchograms on March 24 showed bronchiectasis of lingula and anterior basal segment on the left, and of the middle lobe and medial basal segment on the right. The affected segments of the left lung were resected on April 8. Penicillin (500,000 units) was put into the pleural cavity at operation and streptomycin (0.5 g. b.d.) was given for 17 days post-operatively. Fever up to 101° F. (38.3° C.) was present

for 13 days after operation; this was attributed to atelectasis of the left lower lobe and was not associated with lymphadenopathy or skin rash, and for several weeks she continued to expectorate 2-4 oz. (57-114 ml.) of purulent sputum daily. On April 19 the white-cell count was 9,700 (polymorphs 75%, lymphocytes 22%, monocytes 3%). She was discharged from hospital on May 15.

The patient remained well until November 4, when she developed a right-sided pleuritic pain and had a 4-oz. (114-ml.) haemoptysis. On readmission to hospital her temperature was 101° F. (38.3° C.); there were numerous medium rales at the right base; the urine was normal; a chest radiograph showed patchy consolidation in the right lower zone. On November 5 a blood count showed: Hb, 89%; white cells, 6,800 (polymorphs 76%, lymphocytes 24%); E.S.R., 47 mm. in 1 hour. Chloramphenicol was given for seven days, but slight fever persisted and oxytetracycline was then given for five days. There was complete clearing of the opacities in the chest radiograph. On December 2 the E.S.R. was 26 mm. in 1 hour. A left bronchogram showed complete re-expansion of the left lower lobe. It was decided that she should be readmitted later for resection of the bronchiectatic segments of the right lung.

On December 17 malaise and anorexia developed, followed a day later by jaundice with dark urine and pale stools. The jaundice lasted 14 days. She felt fairly well for about a week and then again felt ill, became feverish, and had a slight sore throat. The fever—100-101° F. (37.8-38.3° C.)—persisted until her readmission to hospital a fortnight later, on January 23, 1953. At first there was no increase in her cough, but three days before admission she started to expectorate 3-4 oz. (85-114 ml.) of blood-stained purulent sputum. On January 22 bilateral purulent conjunctivitis developed. On examination (January 23) she was found to be flushed and ill. Her temperature was 100.8° F. (38.2° C.), pulse rate 90, respirations 28, blood pressure 135/90. There was a severe bilateral blepharitis and conjunctivitis, but no jaundice. Profuse rales and rhonchi were heard at the right base; the sputum was copious, purulent, and blood-stained. The liver was moderately enlarged and tender; the spleen was palpable. There was one firm gland in each axilla. Central nervous system, cardiovascular system, and fundi were normal. The sputum contained no acid-fast bacilli and on culture grew a mixed flora, predominantly *H. influenzae*. Conjunctival swab culture grew *H. influenzae* and *Staph. pyogenes*. The white-cell count was 7,000 (polymorphs 75.5%, lymphocytes 17%, monocytes 7.5%); subsequent counts were 4,400-8,600; E.S.R. 68 mm. in one hour. The urine contained albumin (3.5 parts per 1,000), pus cells, and red cells, and was sterile on culture. A chest radiograph showed patchy consolidation in the right lower zone.

She was given chlortetracycline by mouth, and chlortetracycline eye ointment with mercury oxycyanide irrigations; the conjunctivitis rapidly cleared and the sputum soon became mucoid and greatly reduced in volume. However, fever persisted and her general condition did not improve. On January 28 she complained of pain in the right hip-joint, knees, and left shoulder. A systolic murmur was noted at the pulmonary area. Chlortetracycline was discontinued; sodium salicylate was started in view of the possibility of acute rheumatism, but was stopped after 40 gr. (2.6 g.) had been given, on account of tinnitus and deafness. The temperature fell to normal for the first time, but after 12 hours again began to rise. On January 31 a throat swab did not grow *Str. haemolyticus*; the antistreptolysin titre was less than 50 units per ml. and the differential agglutination titre 64/128; blood culture was sterile. The joint pains subsided, but on February 4 she developed right-sided pleuritic pain with a friction rub, not associated with any increase or change in character of the sputum. On February 6 the temperature was 103.6° F. (39.8° C.); chlortetracycline was restarted.

The temperature settled to normal during the next four days, but there was no improvement in the clinical condition, and severe nausea and vomiting and diarrhoea made it

necessary to stop the chlortetracycline. The serum protein was 6.2 g. per 100 ml. (albumin 2.1 g., globulin 4.1 g.) and blood urea 84 mg. per 100 ml. On February 12 a hard white exudate was seen in the right ocular fundus. There was redness and scaling over the extensor surfaces of the knees and elbows. L.E. cells were demonstrated by incubation of the patient's plasma with normal heparinized marrow. The temperature had risen to 101.4° F. (38.6° C.), and on February 14 cortisone, 200 mg. daily, was started; this was increased to 300 mg. daily on February 17 and she became afebrile. She continued on a maintenance dose of 150 mg. daily. On February 20 the Hb was 59%; white-cell count, 7,200; platelets 240,000 per c.mm.; serum proteins, 6.1 g. per 100 ml. (albumin 1.9 g., globulin 4.2 g.); paper electrophoresis showed increased alpha-2 and gamma-globulins; thymol turbidity, 12 units with flocculation; blood urea, 63 mg. per 100 ml.; urea clearance, 39.8% and 28.4% of normal. On February 28 she developed a perforation of the nasal septum attributed to atrophic rhinitis; W.R. and Kahn test were negative. While on cortisone she put on 5 lb. (2.3 kg.), but there was no hypertension or glycosuria.

On March 1 she developed psychotic symptoms with confusion, visual and auditory hallucinations, and delusions of persecution; this was regarded as a "schizophrenic state developing in a schizoid personality, provoked by cortisone" (Dr. R. F. Tredgold). Cortisone was discontinued on March 4 and the fever gradually mounted again, reaching 103° F. (39.4° C.). Mepacrine, 100 mg. t.d.s., was started on March 13; from this time till her discharge from hospital the temperature fluctuated between normal and 100° F. (37.8° C.). Her general condition and mental state steadily improved, and the mepacrine dose was later reduced to 50 mg. b.d. On April 14 she developed oedema of the legs. At this time the Hb was 52%; serum protein, 5.1 g. per 100 ml. (albumin 1.9 g., globulin 3.2 g.); blood urea, 48 mg. per 100 ml.; the urine contained albumin (4 parts per 1,000), numerous pus cells, red cells, and granular and hyaline casts. On April 28 3 pints (1.7 litres) of whole blood was transfused. On April 30 she had three major epileptic convulsions; the B.P. was 180/100, and there was oedema of the face and legs. The hypertension was controlled with hexamethonium bromide and she had no further fits; hexamethonium bromide was continued until her death. In early June she had a band of vesicles and erythema round the lower right chest; this was thought to be herpes zoster, and it cleared uneventfully. She remained anaemic, but was well enough to be allowed home on July 4. She died at home on July 23, but the immediate cause of death is unknown and there was no necropsy.

Discussion

The combination of hepatitis, albuminuria, hypertension and renal failure, arthralgia, retinal exudates (Wagener, 1946), increased gamma-globulin, and anaemia in an acutely ill febrile patient makes the diagnosis of S.L.E. virtually certain; the diagnosis was confirmed by the finding of L.E. cells. The nephrotic syndrome not uncommonly occurs in S.L.E. (Brenner *et al.*, 1948; Daugherty and Baggenstoss, 1950; Dubois, 1953; Allen, 1955); in these cases wire-loop lesions are present in the glomeruli, and hypertension, too, often occurs in cases with renal involvement. Epileptic convulsions may be the first manifestation of S.L.E. (Russell *et al.*, 1951), and are then due to cerebral angiitis. Convulsions may occur as a result of hypertension from renal involvement or accompanying treatment with cortical steroids. In our case the convulsions were presumably due to hypertensive encephalopathy, probably associated with moderate diastolic hypertension of rapid onset, and cerebral oedema; the convulsions ceased, and did not recur, when the hypertension was controlled with hexamethonium.

This patient is known to have developed severe sulphamide hypersensitivity in March, 1952; the next manifestations of S.L.E. appeared in December, 1952, though in the November there was a persistently high E.S.R. and a slight

fever, but this may have been due to bronchopulmonary infection. She is not known to have been given sulphonamides between March and December, 1952. There were no symptoms or signs before the episodes of sulphonamide hypersensitivity that could have been interpreted as a part of the clinical picture of S.L.E.

It has been pointed out that various drugs, including sulphonamides, may induce acute exacerbations of established S.L.E., and it is reasonable to suppose that such an episode might represent the first overt manifestation of the disease. In this case the administration of sulphonamide appears to have been the trigger which set the disease process in motion, in a patient of the appropriate constitutional type. It is considered that this case provides additional circumstantial evidence for the concept of S.L.E. as a hypersensitivity disease. It is not, of course, suggested that sulphonamides are the only agents which can act in this way, nor that a sulphonamide hypersensitivity reaction necessarily constitutes a part of S.L.E.

Summary and Conclusions

A case of systemic lupus erythematosus is described in which the onset was preceded by severe sulphonamide hypersensitivity.

The evidence that S.L.E. may be an allergic disease is briefly reviewed, and the role of sulphonamides discussed.

This case appears to provide additional circumstantial evidence for the belief that sulphonamides may sometimes be an important aetiological factor in S.L.E.

This patient also developed the nephrotic syndrome and hypertensive encephalopathy; the latter was successfully controlled by hexamethonium.

I wish to thank Dr. Andrew Morland for permission to publish this case.

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PRIMARY CERVICAL DYSTOCIA TREATED WITH CAUDAL ANALGESIA

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Primary cervical dystocia is difficult to place in the classification of abnormal uterine action, and the causes and mechanism are not well understood. It remains one of the more difficult problems in abnormal obstetrics. The treatment usually advised is cervicotomy or caesarean section.

In 1952 Arthur and Johnson reviewed a series of these cases in which the use of caudal analgesia brought about rapid dilatation of the cervix without either incision or manual stretching. The technique, however, is not referred to by Grant (1955) in the publication of a case of annular detachment of the cervix.

The following cases of primary cervical dystocia were all successfully treated by us with caudal analgesia alone.

Case 1

A primigravida aged 21 was admitted to hospital on April 24, 1954, with mild toxæmia of pregnancy. The estimated date of delivery was April 19. The foetal head had been engaged in the pelvis since the 36th week. Vaginal examination at that time had not revealed any abnormality, and the patient was thought to have an adequate pelvis.

On admission to hospital her blood pressure was 140/100. There was pitting oedema of the ankles, puffiness of the face, and swelling of the hands. The same day artificial rupture of the membranes under anaesthesia was attempted to induce labour. It was noted that the cervix was not taken up and that only the tip of one finger could be introduced into the cervical canal with great difficulty. However, the membranes were successfully ruptured with the tip of a Drew Smythe cannula. Labour started within 18 hours of the induction, and by the evening of April 25 the patient was getting strong contractions every three minutes and was very distressed. The sedatives given during the night were heroin and quinalbarbitone.

Vaginal examination after the patient had been in labour 18 hours showed that the cervix was effaced and thinning, with the os dilated one fingerbreadth. The head was very tightly applied to it. The same day she unexpectedly had an eclamptic fit. She was treated with rectal avertin, and this was repeated as necessary. For the rest of that day, although deeply unconscious, the patient continued to have uterine contractions, increasing in strength and frequency. Vaginal examination after 38 hours in labour showed the cervix to be thin and dilated one to two fingerbreadths, with the head so tightly applied to it that it was impossible, even between contractions, to introduce the tip of a finger between the head and the external os, through which a tense caput was bulging.

The strength and regularity of the contractions indicated that there was no incoordinate action of the upper segment, and a diagnosis of primary cervical dystocia was made. Delivery by caesarean section was contemplated, but as the patient's general condition remained satisfactory it was decided to wait for a further 12 hours.

The condition was unchanged after 50 hours in labour. The uterus was contracting strongly and regularly every two to three minutes, and vaginal examination did not show any